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产品名称: 3,3-二(4-羟基苯基)-2(3H)-吡啶酮
产品别名: 酚汀; Oxyphenisatine; Oxyphenisatin

生物活性:						
Description		Oxyphenisatine (Oxyphenisatin) is a laxative. Oxyphenisatin acetate is the pro-drug of oxyphenisatin with anticancer activity.				
In Vitro		Oxyphenisatin has been shown to have antiproliferative activity. Oxyphenisatin acetate (OXY, NSC 59687) is the pro-drug of oxyphenisatin. OXY inhibits the growth of the breast cancer cell lines MCF7, T47D, HS578T, and MDA-MB-468 (IC50=0.8, 0.6, 2.1, 1.8 μM). This effect is associated with selective inhibition of translation accompanied by rapid phosphorylation of the nutrient sensing eIF2α kinases, GCN2 and PERK[1].				
In Vivo		Toxicity studies demonstrate that mice tolerate IP administration of OXY at 300 mg/kg once daily or 200 mg/kg twice daily. Administration of OXY at 300 mg/kg IP once daily for 10 days results in significantly smaller tumors from day 33 to day 52 ^[1] .				
Solvent&Solubility		In Vitro: DMSO : ≥ 28 mg/mL (88.23 mM) * "≥" means soluble, but saturation unknown.				
		Preparing Stock Solutions	Solvent Concentration Mass	1 mg	5 mg	10 mg
			1 mM	3.1512 mL	15.7560 mL	31.5119 mL
			5 mM	0.6302 mL	3.1512 mL	6.3024 mL
			10 mM	0.3151 mL	1.5756 mL	3.1512 mL
*请根据产品在不同溶剂中的溶解度选择合适的溶剂配制储备液 一旦配成溶液，请分装保存，避免反复冻融造成的产品失效。 储备液的保存方式和期限 -80℃, 6 months; -20℃, 1 month。 -80℃ 储存时，请在 6 个月内使用，-20℃ 储存时，请在 1 个月内使用。						
References		[1]. Morrison BL, et al. Oxyphenisatin acetate (NSC 59687) triggers a cell starvation response leading to autophagy, mitochondrial dysfunction, and autocrine TNFα-mediated apoptosis. Cancer Med. 2013 Oct;2(5):687-700.				
实验参考:						
Animal Administration		Mice: When tumors reaches 120 mg, mice are randomized into treatment groups and therapy is initiated. Asimple toxicity assessment to determine tolerability to OXY is conducted by administering single intraperitoneal (IP) doses of compound at 100, 200, and 400 mg/kg. The mice were observed for adverse effects for 14 days postdose[1].				
References		[1]. Morrison BL, et al. Oxyphenisatin acetate (NSC 59687) triggers a cell starvation response leading to autophagy, mitochondrial dysfunction, and autocrine TNFα-mediated apoptosis. Cancer Med. 2013 Oct;2(5):687-700.				